

Acid Base Balance

Dr M A Maleque Molla,
FRCP(Ed), FRCPCH

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Terminology & Definitions

- **pH**-Negative log of the hydrogen ion concentration
 - $\text{pH} = \text{pK} + \log([\text{base}]/[\text{acid}])$
 - *pH represents the hydrogen concentration.*
 - The plasma concentration of the H^+ ion at a pH of 7.4 is 40 nmol/L.
 - Neutral pH is 7 at temp 25° C & 6.8 at 37°C (Water)
 - Normal pH of body fluids:
 - Arterial blood is 7.4, **Range 7.35-7.45**
 - Venous blood and interstitial fluid is 7.35
 - Intracellular fluid pH 7.0
 - *Most important pH for the body is intracellular pH*
 - *pH compatible with life 6.8-7.8*

Terminology & Definitions (Cont..)

- **Acid** - Any substance that can donate proton (H^+)
e.g. H_2CO_3 , NH_3 , HCL ,
- **Base**- Any substance that can accept proton
e.g. HCO_3 , PO_4 , protein
- **Acidemia**- $pH < \text{normal range}(7.35-7.45)$. i.e. H^+ in the blood above normal range
- **Alkalemia**- $pH > \text{normal}(7.35-7.45)$. i.e. H^+ in the blood less than normal range
- **Acidosis** & **alkalosis** is a pathologic process that causes an increase or decrease in the hydrogen ion concentration,
- **Buffers** are substances that has an ability to minimise the change in pH that occurs when acids or bases are added to the body

Acid-Base Balance

Def: Maintenance of a normal balance between production and excretion of acid or alkali by the body, resulting in a stable concentration of H^+ in body fluids.

Acid load in the body

- An adult normally produces 1-2 mEq/kg/24 hr of hydrogen ions.
- Children produce 2-3 mEq/kg/24 hr of hydrogen ions.
- There are 2 types of acids contribute to the daily acid load;
 - Volatile acid-Carbonic acid (H_2CO_3)
 - Nonvolatile acids e.g. H_2SO_4 , Organic acids, phosphoric acid

Acid load in the body

The 3 principal sources of acid (H^+):

- Dietary protein metabolism,
- Incomplete metabolism of carbohydrates and fat:
 - Incomplete glucose metabolism can produce lactic acid,
 - Incomplete triglyceride metabolism can produce keto acids, e.g. β -hydroxybutyric acid and acetoacetic acid.
- Losses of bicarbonate in the stool is one of the major source of acid production
 - For loss of each bicarbonate molecule in the stool, there is addition of 1 hydrogen ion in the body.
- Normally acid production is minimal but may increase dramatically in some disease e.g. diarrhea.
- *In order to maintain acid-base homeostasis, acid production must balance the neutralization or excretion.*

Daily H⁺ Balance

	INPUT Mmol/day		OUTPUT Mmol/day
Volatile:			
• CO ₂	13000	Lungs	13000
• Lactate	1500	Liver, Kidney	1500
Nonvolatile:			
• Protein(SO ₄)	45	Titratable acid	30
• Phosphate(PO ₄)	30	Ammonia	40
•Others	12		

Why acid-base balance needed?

- Regulation of normal pH is necessary because;
 - For the proper functioning of cellular enzymes and other metabolic processes.
 - Chronic derangements in acid-base status interfere with normal growth and development of child.
 - Acute, severe changes in pH can be fatal.

How acid-base balance is regulated?

- Body strictly maintains pH in a range from **7.35-7.45**
- Changes in H^+ concentration are prevented by body's buffering system;
 1. *Cellular buffers*
 2. *Lungs*
 3. *Kidneys,*

1. Intracellular and extracellular buffers

- Important cellular buffers:
 1. Bicarbonate/carbonic acid buffer
 2. Non bicarbonate buffers
 - a) Protein buffers
 - b) Phosphate buffers
 - c) Bone

Blood Buffer system

1. Bicarbonate/carbonic acid buffer.

- Most abundant buffer in ECF
- Function almost instantaneously within seconds.

- Cells utilizing O_2 & produces CO_2



- CO_2 enter RBC & combines with water to form carbonic acid(H_2CO_3), which dissociates to form H^+ and HCO_3^- :



- HCO_3^- is pumped out to plasma
- At the alveoli, HCO_3^- re-enter the RBC & the above equation is driven to the left, re-producing CO_2 & eliminated by the lung.

Bicarbonate/carbonic acid buffer System

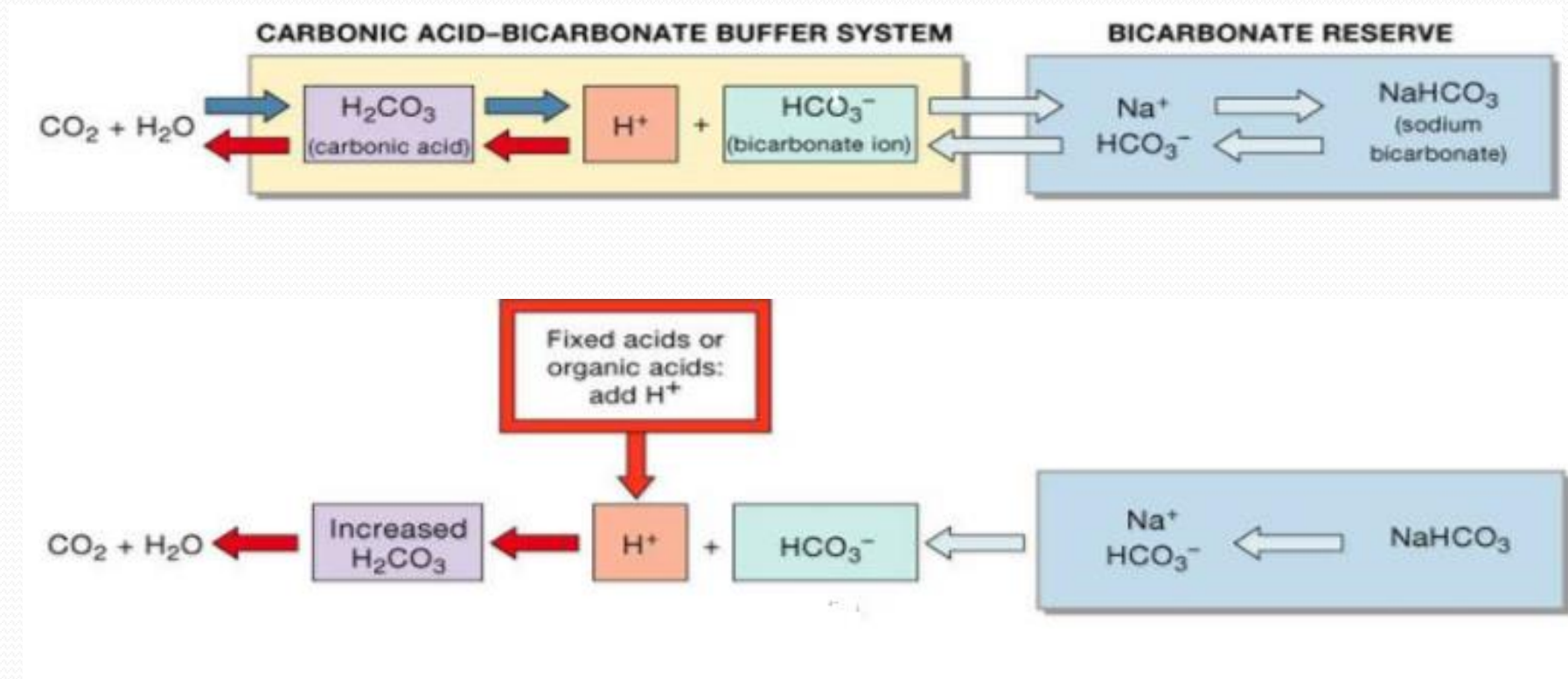


Fig. Response to acidosis

Buffer system (Cont..)

2. Non bicarbonate buffers

a) Protein buffers:

- Extracellular proteins, mostly albumin.
- Intracellular proteins, including hemoglobin.
- Proteins are effective buffers, due to the presence of the amino acid ***histidine***, which can bind or release hydrogen ions.
- Protein can buffers both hydrogen ions(H^+) and carbon dioxide(CO_2).

Buffer system (Cont..)

b) Phosphate Buffers

- Phosphate is an intracellular buffer & important buffer in the Urine
- Has a major role in the elimination of H^+ via the kidney
- Can bind up to 3 hydrogen molecules.
- At a physiologic pH, most phosphate exists as either HPO_4^{2-} or $H_2PO_4^{1-}$

Buffer system (Cont..)

c) **Bone**

- Bone composed of compounds such as sodium bicarbonate and calcium carbonate
- dissolution of bone releases base & can buffer an acid load.
- In contrast, bone formation helps to buffer excess base.

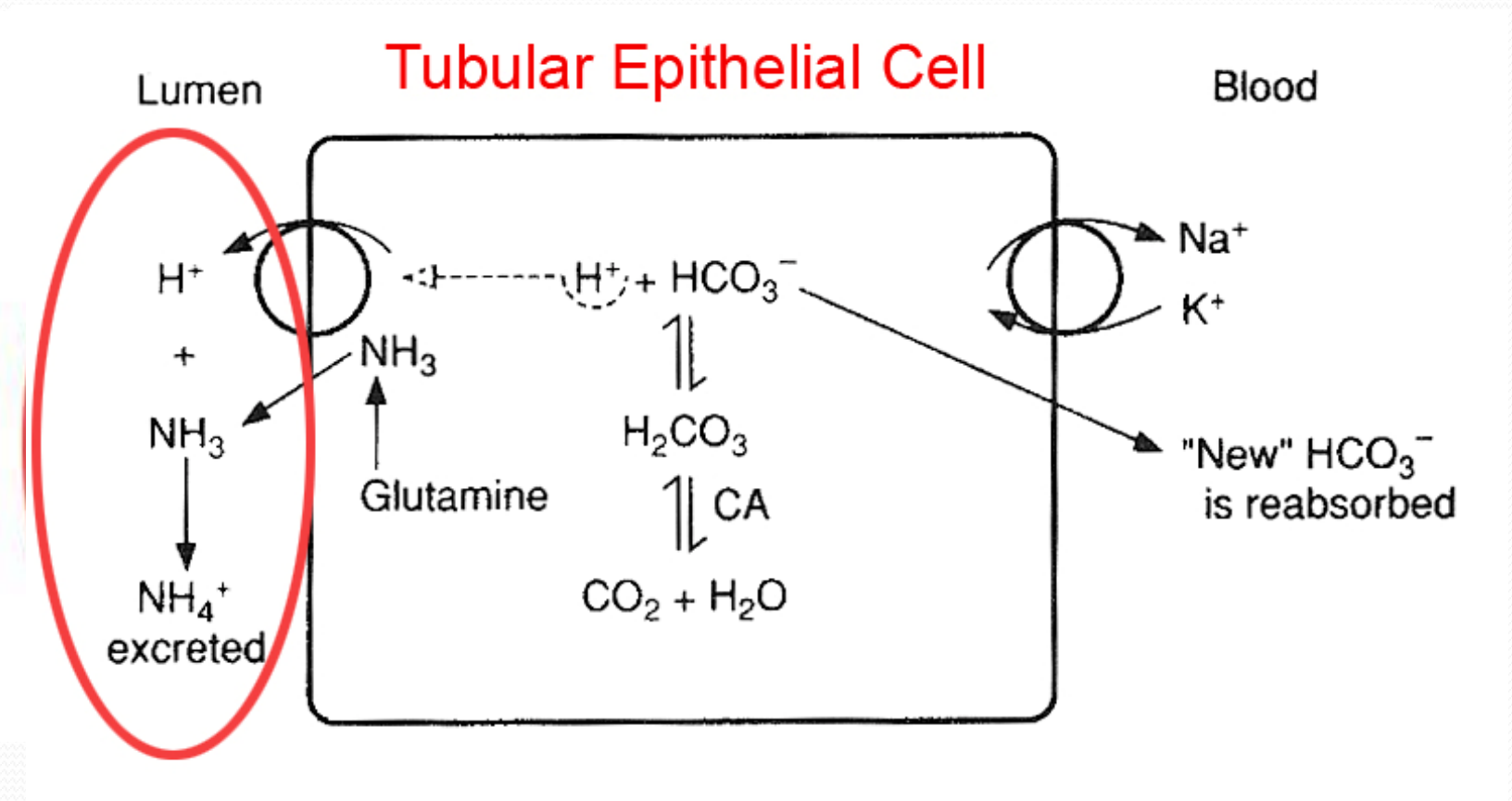
2. Respiratory Control mechanisms on pH

- Works within minutes - maximal in 12-24 hours
- During a simple metabolic disorder, there is respiratory compensation.
- **In case of metabolic acidosis:**
 - There is \uparrow H^+ & \downarrow pH in the ECF.
 - Major source of H^+ ion in the body is CO_2 :
 - Excess H^+ & CO_2 act directly on respiratory centers, increase rate & depth of respiration & eliminate CO_2 & \uparrow pH
- **In case of metabolic alkalosis**(pH>7.45):
 - respiratory center is inhibited
 - retention of CO_2 , \uparrow H^+ production leading to \downarrow pH.

3. Renal Control Mechanisms on pH

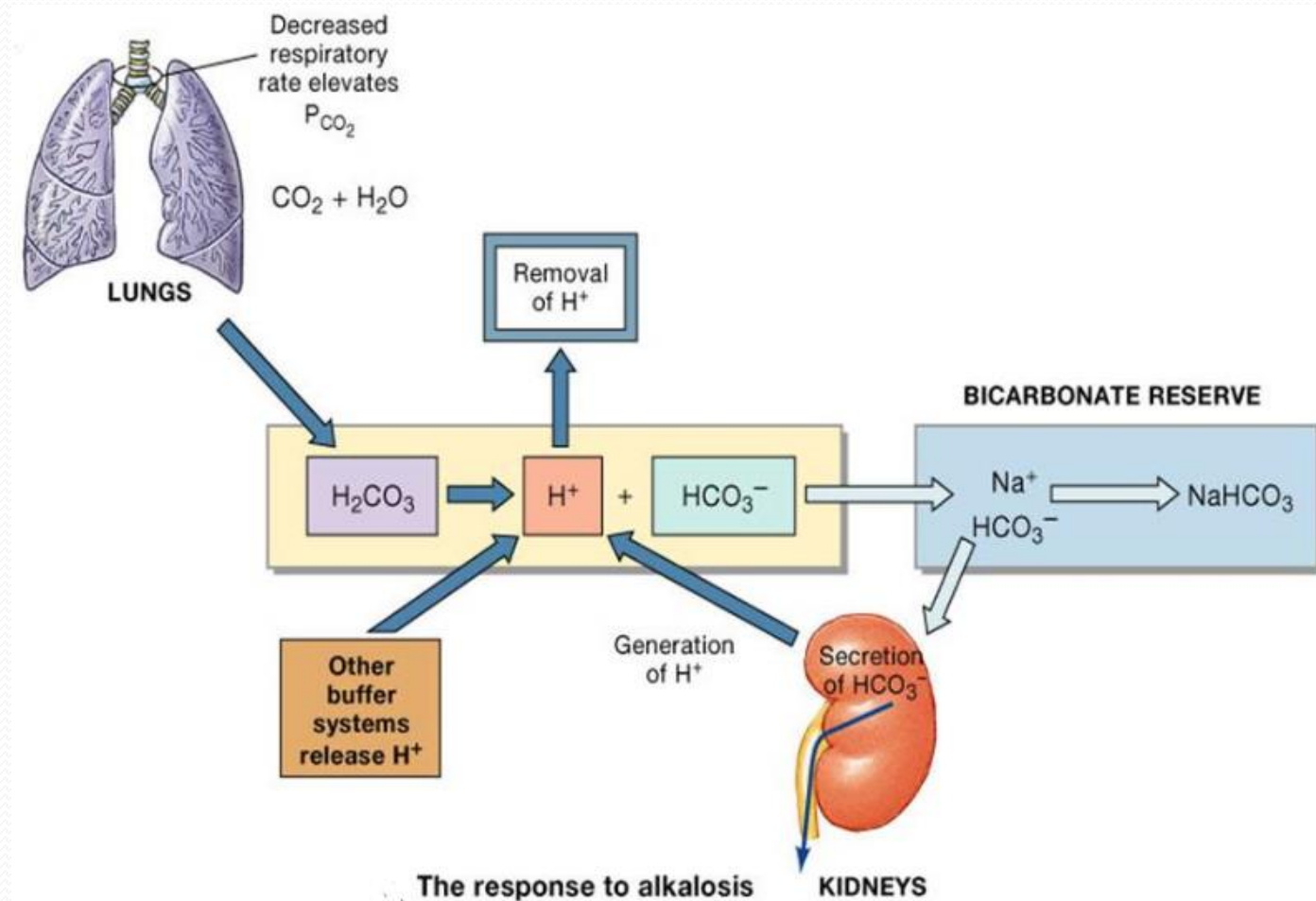
- Kidney takes several hours to days to act & restore pH to, or close to normal.
- Regulates plasma bicarbonate & pH by 3 ways;
 1. Regeneration & reabsorption of filtered HCO_3^- ;
 - Kidneys handles around 4000 mEq of HCO_3^- daily.
 - Almost all HCO_3^- are absorbed in renal tubules.
 2. Formation of titratable acids H_2PO_4^- in the distal tubule & excrete H^+
 3. Excretion of NH_4^+ in the distal tubule and excrete H^+

3. Renal Control Mechanisms on pH(cont..)



Reabsorption of HCO₃ and excretion of H⁺ by kidney

Respiratory & Renal control on Ph





Disorder in acid base balance

Disorder in acid base balance

- ACIDOSIS ($\text{pH} < 7.35$)
 - Metabolic acidosis
 - Respiratory acidosis
 - Mixed acidosis(Combined)
- ALKALOSIS($\text{pH} > 7.45$)
 - Metabolic alkalosis
 - Respiratory alkalosis

Metabolic acidosis

Def: $\text{pH} < 7.35$ due to $\uparrow \text{H}^+$ concentration.

Causes:

- Exogenous source
- Endogenous \uparrow production;
 - DKA,
 - Organic acidemias e.g. Methylmalonic acidemia, propionic acidemia.
 - Lactic acidosis.
- Inadequate excretion of acid; Renal failure
- Excessive loss of HCO_3^- - Gastroenteritis

Metabolic acidosis(cont..)

Compensation:

- Buffered by;
 - ECF - HCO_3
 - ICF – Protein(Hb), Po_4 , bone
- Respiratory compensation:
 - ↑respiration in rate & depth,
 - ↑ alveolar ventilation → ↓ PCO_2
- Renal compensation:
 - ↑Ammonia production + H^+ excretion
 - ↑ HCO_3 reabsorption

Metabolic acidosis(cont..)

Clinical features:

- **Hyperventilation.**
- **Decrease cardiac function:** pH <7.20, impaired cardiac contractility and an increased risk of arrhythmias
- **Hypotension.**
- **Pulmonary oedema** leading to hypoxia.
- **Lethargy and coma:** Severe acidemia impairs brain metabolism
- The acute metabolic effects:
 - insulin resistance,
 - increased protein degradation,
 - reduced ATP synthesis

Metabolic acidosis (cont..)

- Lab:
 - BUN, serum creatinine, serum glucose, urinalysis, and serum electrolytes
 - Plasma anion gap.
- Acid base status : **pH <7.35, ↓ PCO₂ ↓ HCO₃.**
- Treatment mainly treating underlying causes.
- Bicarbonate therapy may be needed.

Anion gap

Def: Calculated difference between anion & cation electrolytes.

■ Anion gap metabolic acidosis (anion gap > 16):

➤ **'MUDPILES'** = **M**ethanol intoxication, **U**remia, **D**iabetic or alcoholic ketoacidosis, **P**araldehyde, **I**soniazid or **I**ron overdose, **L**actic acid, **E**thylene glycol intoxication, **S**alicylate intoxication

■ Normal or non-anion gap acidosis (anion gap 12 ± 4)

- GI loss of HCO_3^- (diarrhea)
- Renal loss of HCO_3^- e.g Renal tubular acidosis
- Compensation for respiratory alkalosis
- Ureteral diversion

■ Decrease anion Gap: < 8

- Reduction in a major plasma protein such as albumin (renal loss).
- Hyperlipidemias and other less common causes.

Respiratory acidosis

- Inadequate elimination of CO₂ due to decrease alveolar ventilation.
- **Causes:**
 - Airway obstruction
 - Neuromuscular disease
 - Sedative over dose
- **Compensation in 2 phase:**
 - Cell buffering by protein and Hb:
$$\text{H}_2\text{CO}_3 + \text{Hb}^- \rightarrow \text{HHb} + \text{HCO}_3^-$$
 - Renal compensation by reabsorption of more HCO₃.
- **Acid base status:** pH <7.35, ↑PCO₂, Normal or ↑HCO₃
- **Treatment:**
 - Respiratory support e.g. Mechanical ventilation in severe cases.

Respiratory alkalosis

- Inappropriate reduction of CO₂ concentration.

Causes:

- Usually due to hyperventilation:
 - Early pneumonia, asthma.
 - Iatrogenic-Pt is on mechanical ventilator .
 - Psychogenic.
 - Drugs like Salicylate poisoning.
- Respiratory alkalosis without hyperventilation:
 - Receiving extracorporeal membrane oxygenation, hemodialysis.
- **Compensation:** mainly renal.
 - Decrease in renal excretion of H⁺
 - Cell buffering by moving hydrogen ions from the cells into the extracellular fluid.

Respiratory alkalosis

CLINICAL FEATURE

- Acute respiratory alkalosis:
 - Chest tightness, palpitations,
 - Lightheadedness,
 - Circumoral numbness,
 - Paresthesias of the extremities.
 - Tetany, seizures, muscle cramps, and syncope are less common
- Chronic one is usually asymptomatic

DIAGNOSIS

Lab: depend upon the history & physical findings

Acid base status: **pH >7.45, ↓ PCO₂, ↓ HCO₃**

Respiratory alkalosis(cont..)

Management:

- Underlying cause should be treated
- If on mechanical ventilator, setting should be adjusted
- Psychogenic hyperventilation may benefit from
 - reassurance
 - benzodiazepines.
 - Re-breathing into a paper bag.

Metabolic alkalosis

- Decrease acid below the normal range
- Causes: divided into 2 categories on the basis of urinary chloride level
 1. *Chloride responsive (Urinary chloride <15 mEq/l).*
 - Excessive loss of H^+ e.g. vomiting, Nasogastric suction.
 - Diuretics (loop or thiazide).
 - Decrease serum Cl^- .
 - Contraction of ECF.
 - Cystic fibrosis.
 - Chloride-losing diarrhea.
 - Post-hypercapnia.
 2. *Chloride resistant (Urinary chloride >20).*
 - Hyperaldosteronism
 - Cushing syndrome.
 - Bartter's syndrome.
 - Severe hypokalemia

Metabolic alkalosis(cont.)

- Clinical feature:
 - The symptoms are often related to the underlying disease and associated electrolyte disturbances.
 - Symptoms related to volume depletion, such as thirst and lethargy.
 - Chloride-unresponsive causes may have symptoms related to hypertension
 - General feature includes muscle cramps, tetany
- Compensation:
 - Respiratory : ↓Respiratory rate, ↑ PCO₂
 - Renal: loss of HCO₃ in urine
- Acid base status: **pH >7.45 , Normal or ↑PCO₂, ↑HCO₃**

Metabolic alkalosis(cont.)

Management;

- Depends on the severity and the underlying cause.
- Mild ($\text{HCO}_3^- < 32$): treatment is often unnecessary
- Moderate or severe metabolic alkalosis;
 - Treat underlying cause e.g. if receiving diuretic add potassium sparing one.
 - Supplement of sodium chloride and potassium chloride to correct the *volume deficit* and the potassium deficit.

Mixed acid base disorder

- When there is more than one acid base disturbance present simultaneously.
- It is suspected when;
 - The expected compensatory response does not occur.
 - Compensatory response occurs, but level of compensation is inadequate or too extreme.
 - Whenever the PCO_2 and $[\text{HCO}_3^-]$ becomes abnormal in the opposite direction.
 - pH is normal but PCO_2 or HCO_3^- is abnormal.
 - In anion gap metabolic acidosis, if the change in bicarbonate level is not proportional to the change of the anion gap.
 - In simple acid base disorders, the compensatory response should never return the pH to normal. If that happens, suspect a mixed disorder.



Diagnosis of acid base disorders

- Clinical history & Examination
- Evaluation of an arterial blood gas sample.

Terminology used in blood gas analysis

PCO ₂	44.6 mmHg
Pat. ID	
Last Name	
First Name	
Temperature	37.0 °C
FIO ₂	0.21
Sample type	Blood
Blood type	Unknown
Age (A/F)	> 1 year
pH	7.420
PO ₂	78.7 mmHg (-)
BE	3.4 mmol/L
cHCO ₃ ⁻	28.2 mmol/L
tHb	8.5 g/dL (-)
SO ₂	96.2 %
O ₂ Hb	94.6 % (-)
COHb	1.0 %
HHb	3.7 %
MetHb	0.7 %

- **PaCO₂**-Partial pressure of carbon dioxide in arterial blood.
N 35-45 mmHg
- **pH**-Negative logarithm of hydrogen ion concentration . N 7.35-7.45
- **PaO₂**- Partial pressure of oxygen in arterial blood. N= 80-100 mmHg
- **BE**- calculate the quantity of Acid or Alkali required to return the plasma in-vitro to a normal pH under standard conditions.
N=0 (-2 to +2 mmol/l)
- **HCO₃**-Bicarbonate concentration in the serum in mmol/l . N= 22-26 mmol/L
- **SO₂**- Oxygen saturation >92%

How blood gas can be analyzed?

Blood gas can be analyzed by automated machine using following types of blood samples:

- Arterial Blood Gas (ABG)
- Venous Blood Gas (VBG)
- Capillary Blood Gas (CBG)

Normal values

	ABG	VBG	CBG
pH	7.35-7.45	7.25-7.35	7.35 – 7.45
PCO ₂ (mmHg)	35-45	41-51	35 – 48
PO ₂ (mmHg)	80-100	35-40	80-100
HCO ₃ (mmol/L)	22-26	22-26	22 – 27
BE (mmol/L)	± 2	± 2	±2

Interpretation of blood gas

RADIOMETER ABL800 BASIC

ABL800 BASIC PICU
PATIENT REPORT Syringe - S 195uL 01:55 PM 11/3/2014
Sample # 2175

Identifications

Patient ID
Patient Last Name
Patient First Name
Sample type Not specified
T 37.0 °C

Blood Gas Values

pH	7.109		[-]
pCO ₂	9.4	mmHg	[-]
pO ₂	93.6	mmHg	[-]

Oximetry Values

ctHb	10.8	g/dL	[-]
sO ₂	96.1	%	[-]
FO ₂ Hb _e	95.4	%	[-]
FHHb _e	3.8	%	[-]

Electrolyte Values

cK ⁺	5.3	mmol/L	[-]
cNa ⁺	130	mmol/L	[-]

Temperature Corrected Values

pH(T)	7.109	
pCO ₂ (T)	9.4	mmHg
pO ₂ (T)	93.6	mmHg

Oxygen Status

ctO _{2e}	14.5	Vol%
p50 _e	30.48	mmHg

Acid Base Status

cBase(Ecf) _C	-25.7	mmol/L
cHCO ₃ ⁻ (P,st) _C	6.5	mmol/L



- pH 7.109
 - pCO₂ 9.4 mmHg
 - pO₂ 93.6 mmHg
 - sO₂ 96.1%
 - Na 130 mmol/l
 - K 5.3 mmol/l
 - HCO₃ 6.5 mol/l
 - BE -25.7 mmol/l
-
- What type of acid base disorder the child has?

Stepwise interpretation of blood gas

- Step I: Acidosis or Alkalosis



1. Look for pH

- Normal pH 7.35-7.45
 - pH < 7.35 Acidosis
 - pH > 7.45 Alkalosis

- *Remember: an acidosis or alkalosis may be present even if the pH is in the normal range*

Stepwise interpretation of blood gas

- Step 2: Respiratory or Metabolic



- Look for PCO_2 and HCO_3^-

- \downarrow pH < 7.35 \downarrow pCO_2 & \downarrow HCO_3^- - Metabolic acidosis
- \uparrow pH > 7.45 \uparrow HCO_3^- & \uparrow pCO_2 - Metabolic Alkalosis
- \downarrow pH < 7.35 \uparrow pCO_2 & N or \uparrow HCO_3^- - Respiratory acidosis
- \uparrow pH > 7.45 \downarrow pCO_2 & \downarrow HCO_3^- - Respiratory Alkalosis
- In metabolic disorders the pH and PaCO_2 change in the same direction
- In respiratory disorders, the pH and PaCO_2 change in *opposite* directions.

Stepwise interpretation of blood gas

Step 3: If respiratory acidosis

Acute or Chronic?



- Look for bicarbonate



- Normal slightly raised HCO_3 – Acute Respiratory acidosis
- High HCO_3 – Chronic Respiratory acidosis

Stepwise interpretation of blood gas

Step 4: For metabolic acidosis



Look for anion gap

$$\text{Anion gap} = (Na^{+} + K^{+}) - (Cl^{-} + HCO_{3}^{-}).$$

- Anion gap 12 ± 4 mmol/l = Normal or non-anion gap acidosis
- Anion gap > 16 mmol/l- Anion gap metabolic acidosis

Stepwise interpretation of blood gas

Step 5. Determine whether other metabolic disturbances co-exist with an anion gap acidosis



Measure corrected bicarbonate.

Corrected $\text{HCO}_3^- = \text{measured } \text{HCO}_3^- + (\text{anion gap} - 12)$

- If the corrected $\text{HCO}_3^- =$
 - 22-26 mEq/l- pure anion gap metabolic acidosis
 - >26 mEq/l -a metabolic alkalosis co-exists.
 - 22mEq/l- then additional non-gap acidosis co-exists with anion gap acidosis.

Stepwise interpretation of blood gas

Step 6: Look for compensatory response

- The body's attempt to return the acid/base status to normal.



Metabolic Acidosis	Primary change ↓ HCO_3^-	Compensatory response ↓ PCO_2
Respiratory Acidosis	Primary change ↑ PCO_2	Compensatory response ↑ HCO_3^-
Metabolic alkalosis	Primary change ↑ HCO_3^-	Compensatory response ↑ PCO_2
Respiratory alkalosis	Primary change ↓ PCO_2	Compensatory response ↓ HCO_3^-

Compensatory response

Metabolic acidosis:

- *Respiratory & renal compensation:*
 1. Immediate buffering by HCO_3^- in ECF.
 2. Respiratory compensation in minutes :
 - By ↑ Rate and depth of respiration.
 - For each 1.2 mmol decrease in HCO_3^- , a 1 mmHg drop of PaCO_2 .
 3. Tissue phase (2 h): Entry of H^+ into cells accounts for 60% of rapid buffering of poorly permeable acids (HCl or H_2SO_4).
 4. Renal compensation hours to days :
 - by excretion of acid or
 - by conservation of more HCO_3^- .

Compensatory response (cont..)

Respiratory Acidosis:

- *Mainly renal compensation.*
- ↑ in PCO_2 leads to an elevation of $[\text{H}^+]$ concentration.
- Body responds by trying to increase the plasma $[\text{HCO}_3^-]$.
- This is accomplished via two mechanisms;
 - a) Rapid cell buffering
 - b) Increase in net acid excretion by the kidney.

Compensatory response (cont..)

Acute respiratory acidosis:

- The $\uparrow\text{CO}_2$ levels lead to an increase in carbonic acids, (H_2CO_3).
- H_2CO_3 cannot be buffered by HCO_3^- in the extracellular fluid.
- Therefore bicarbonate levels do not fall in respiratory acidosis.
- Intracellular buffering of H_2CO_3 by hemoglobin and proteins.

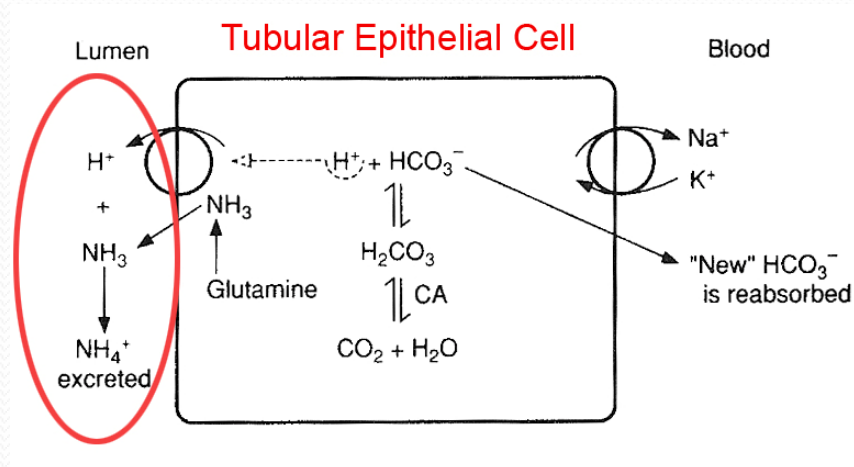


- There is an increase in the plasma $[\text{HCO}_3^-]$, averaging 1 meq/L for every 10 mmHg rise in the PCO_2 .

Compensatory response (cont..)

Chronic Respiratory Acidosis:

- Compensation mainly Renal
- Compensation complete after 3-5 days
- Persistent \uparrow PCO₂ stimulates increased excretion of titratable acid and ammonium, resulting in the addition of new HCO₃⁻ to the extracellular fluid.



- A 3.5 meq/L increase in the plasma HCO₃⁻ concentration for every 10 mmHg increase in the PCO₂.

Compensatory response (cont..)

Metabolic Alkalosis:

- Mainly Respiratory compensation.
- ↑ in $[\text{HCO}_3^-]$ above the normal range, and subsequently a ↓ in hydrogen ion concentration
- **Compensation by ↓ respiratory** rate thus ↑ in PCO_2 & H^+ concentration.
- *On average the $p\text{CO}_2$ rises 0.7 mmHg for every 1.0 meq/L increment in the plasma $[\text{HCO}_3^-]$.*

Compensatory response (cont..)

Respiratory Alkalosis:

- Mainly renal compensation.
- There is \uparrow in the frequency of alveolar ventilation & increase in minute ventilation.
- This leads to a \downarrow PCO_2 & thus \downarrow in H^+ concentration.
- Body responds by trying to reduce the plasma $[\text{HCO}_3^-]$ to match the reduction in PCO_2 and thus maintain the ratio by;
 - 1) Rapid cell buffering
 - 2) A decrease in net renal **acid** excretion.

Compensatory response (cont..)

- **Acute respiratory alkalosis:** >10 min.
 - Compensation by rapid cell buffering by protein.
 - *Every 10 mmHg decrease in the PCO_2 , there is a 2meq/L decrease in the plasma HCO_3^- concentration.*
- **Chronic respiratory alkalosis:**
 - Compensation mainly renal & takes >2-6 hr
 - Kidney will respond by:
 - lowering H^+ secretion,
 - excretion of titratable acids,
 - ammonium production and ammonium excretion,
 - increase in HCO_3^- excreted due to decreased reabsorption of filtered HCO_3^- .
 - *For every 10 mmHg reduction in PCO_2 . there is 4 meq/L reduction in plasma $[HCO_3^-]$*

Compensatory responses and their mechanisms.

Primary disorder	Primary Chemical change	Compensatory response	Compensatory Mechanism
Metabolic Acidosis	$\downarrow \text{HCO}_3^-$	$\downarrow \text{PCO}_2$	Hyperventilation
Metabolic Alkalosis	$\uparrow \text{HCO}_3^-$	$\uparrow \text{PCO}_2$	Hypoventilation
Respiratory Acidosis	$\uparrow \text{PCO}_2$	$\uparrow \text{HCO}_3^-$	
Acute			Intracellular Buffering
Chronic			Renal Generation of HCO_3^-
Respiratory Alkalosis	$\downarrow \text{PCO}_2$	$\downarrow \text{HCO}_3^-$	
Acute			Intracellular Buffering
Chronic			Renal Generation of HCO_3^-

Expected level of compensation

Primary disorder	Expected level of compensation
Metabolic Acidosis	<ul style="list-style-type: none"> • $PCO_2 = (1.5 \times [HCO_3^-]) + 8 \pm 2$ • For each 1 mEq/L decrease in serum $[HCO_3^-]$, 1.2 mm Hg decreases of P_{CO_2}.
Metabolic Alkalosis	<ul style="list-style-type: none"> • $PCO_2 = (0.9 \times [HCO_3^-]) + 16 \pm 2$ • P_{CO_2} increases by 7 mm Hg for each 10-mEq/L increase in serum $[HCO_3^-]$
Respiratory Acidosis:	
• Acute	$\uparrow[HCO_3^-]$ by 1 mEq/L for every 10 mm Hg ΔPCO_2
• Chronic	$\uparrow[HCO_3^-]$ by 3.5 mEq/L for every 10 mm Hg ΔPCO_2
Respiratory Alkalosis:	
• Acute	$\downarrow[HCO_3^-]$ by 2 mEq/L for every 10 mm Hg ΔPCO_2
• Chronic	$\downarrow[HCO_3^-]$ by 4 mEq/L for every 10 mm Hg ΔPCO_2



ABG Interpretation 1

- pH = 7.202
- PaCO₂ = 19.8
- PaO₂ = 86.6
- HCO₃⁻ = 7.4
- BE = -18.
- Sat = 91.5
- Hb = 12
- Na⁺ = 153
- K⁺ = 3.4
- Cl⁻ = 123

• Metabolic acidosis

• ? Anion gap

$$\text{Anion gap} = (153 + 3.4) - (123 - 7.4) = 26.22$$

Δ Anion gap metabolic acidosis

Adequate Compensation?

$$\text{PCO}_2 = (1.5 \times [\text{HCO}_3^-]) + 8 \pm 2$$

$$= (1.5 \times 7.4) + 8 = 19.1 \pm 2 = 17.1 - 21.1$$

ABG Interpretation 2

ABG

- pH 7.31
- pCO₂ 33 mmHg
- HCO₃ 16 mmol/l
- PO₂ 93 mmHg
- Na⁺ 134, K⁺ 2.9, Cl⁻ 108, BUN 31, Cr 1.5.

Δ Metabolic Acidosis

- ?Anion gap
- $(134 + 2.9) - (108 + 16) = 12.9$

Δ Non anion gap metabolic acidosis

- Compensation: $PCO_2 = (1.5 \times 16) + 8 = 32$

ABG Interpretation 3

- pH 7.29
- PCO₂ 64.3 mmHg
- PO₂ 84.6 mmHg
- HCO₃ 26.2 mmol/l
- BE -2 mmol/l

Respiratory acidosis

Acute or chronic?

- 1 mmol rise of HCO₃ for every 10 mmHg rise of PCO₂ in acute Rp acidosis
- $64.3 - 40 = 24.3$. So HCO₃ should be $24 + 2.4 = 26.4$
- Δ Acute respiratory acidosis

ABG Interpretation 4

- pH 7.39
- PCO₂ 68.5 mmHg
- PO₂ 84.6 mmHg
- HCO₃ 32.2 mmol/l
- BE +8 mmol/l

Respiratory acidosis

Acute or chronic?

Pco₂ raise: $68.5 - 40 = 28.5$.

Rise HCO₃: $32.2 - 24 = 8.2$ mmol

Δ Chronic respiratory acidosis

ABG Interpretation 5

pH	7.489
PCo ₂	24.9 mmHg
PO ₂	72.4 mmHg
SAT	96.4%
HCO ₃	21.6 mmol/l

- Respiratory alkalosis
- ?Acute or chronic
- Fall of Pco₂=40-24.9=15.1
- Acute -Fall of HCO₃ should be=1.5x2=3. i.e. 24-3=21 mmol/l
- In chronic- Fall of HCO₃ should be=1.5x4=6, i.e. 18 mmol/l
- Acute Respiratory alkalosis

ABG Interpretation 6

- pH = 7.490
- PaCO₂ = 47.0
- PaO₂ = 58.0
- HCO₃⁻ = 34.8
- BE = 10.2
- Sat = 88.9
- Hb = 18.3

Δ Metabolic alkalosis

?Is compensation adequate

Rise of HCO₃ = 34.8-24=10.8

PCo₂ should rise=0.7 x 10.8=7.56, i.e. 40+7.56= 47.57

ABG Interpretation 7

• pH	6.90 ↓
• Pco ₂	79.3 mmHg ↑
• PO ₂	25.2 mmHg
• BE	-15.8 mmol/l ↓
• HCO ₃	12 mmol/l
• SAT	31.5%
• Na	136 mmol/l
• K	4.1 mmol/l
• Chloride	120 mmol/l

- ? Respiratory or metabolic acidosis
- pH is low, Pco₂ is high and HCO₃⁻ is low.
Compensating parameters are in opposite direction.
- Combined respiratory and Metabolic acidosis.

THANKS

